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ARTERIAL INJURIES

Early Diagnosis and Treatment

by

The Vascular Injuries Sub-Committee of the M.R.C. War Wounds Committee

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INTRODUCTION

Wounds of the main arteries of the limbs and neck are not uncommon in warfare and are important not only because they may cause severe hæmorrhage, but also because, by interruption of the blood supply, they often lead to ischæmia of the distal parts of the body. This ischæmia may be followed by a spread of infection (anærobic or ærobic, the former sometimes unbelievably rapid), by gangrene or by varying degrees of muscular, nervous or cerebral degeneration. In many instances, irreparable damage to the limb necessitates a primary amputation, but in others the artery is the only important structure injured, though adjacent nerves are frequently involved. *This Memorandum is intended for the guidance of those who have had only limited previous experience of the early treatment of arterial wounds.*

MORBID ANATOMY

In warfare most vascular injuries are produced by high velocity projectiles, though some are caused by stabbing (e.g., by bayonet); a peculiar feature of the former is that the artery may be damaged by the "lateral concussive effect" of the projectile, even though it does not touch the vessel; moreover, if an artery is wounded, its wall will be damaged to some extent in the area contiguous to the wound.

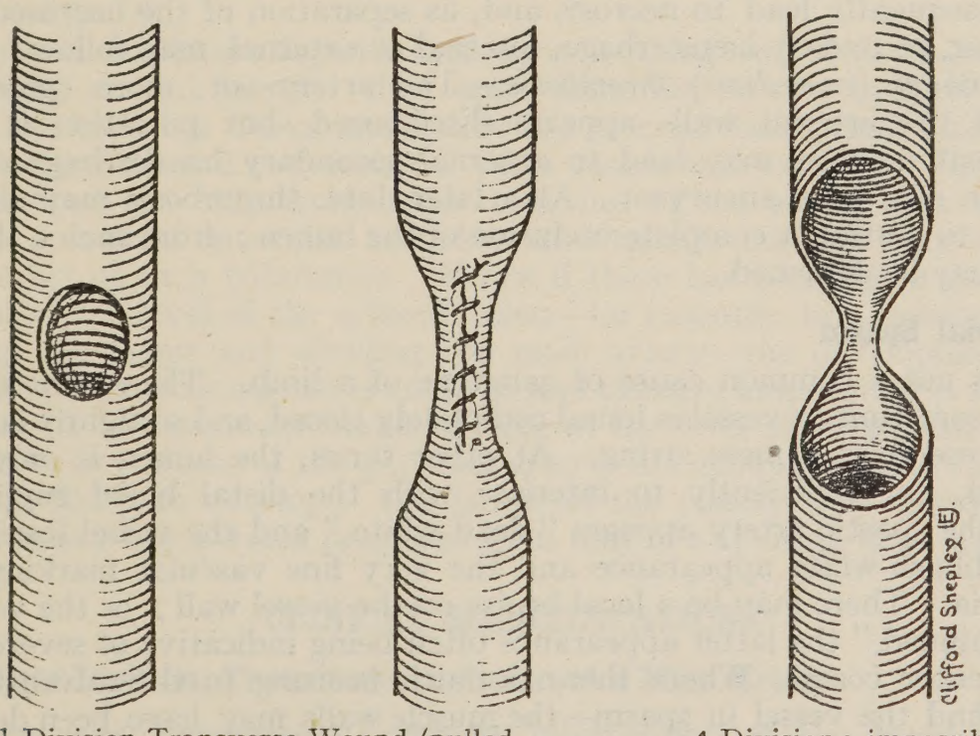
A. Complete Division of an Artery

Both ends of the artery retract within their sheaths, and although usually hæmorrhage is at first profuse, it often ceases spontaneously. The cessation of the hæmorrhage depends upon the free retraction of both ends of the severed vessel, and should this be prevented by the anchoring effect of a branch in close proximity to either end, hæmorrhage is likely to persist.

B. Partial Division of an Artery

Owing to the retraction of the elastic coat of the severed portion of the arterial wall, a transverse wound tends to enlarge, to a greater extent in the long axis of the vessel than in the transverse; thus the opening assumes an oval shape (Fig. 1). In so-called "four-fifths" division, the oval shape may be so

FIG. 1



¼ Division Transverse Wound (pulled into Circle by Retraction of Muscular and Elastic Coats); may sometimes be sutured longitudinally (see pp. 16 and 20), but is apt to produce considerable narrowing.

⅘ Division; impossible to suture except by axial anastomosis.

extreme as to give the appearance of complete division, and the proximal and distal segments of the vessel form a re-entrant angle ; hæmorrhage is profuse, and may be continuous or repeated. If the entry and/or exit wounds in the overlying soft tissues are large, the hæmorrhage is external. If the entry and/or exit wounds in the soft tissues are small, the imbrication of the various muscular and fascial planes provides an efficient barrier to the escape of blood externally, and the hæmorrhage will be internal ; the result is the formation of an arterial hæmatoma, which may subsequently become a false aneurysm, or, if the vein is wounded at the same time, an aneurysmal varix or varicose aneurysm may be produced : in the former, the injured artery and vein communicate directly ; in the latter, they are associated with a false sac.

It is important to realise that in all such cases there is a stream of blood flowing along the main vessel, past the arterial wound, into the distal region of the body, and that the volume of this is often sufficient to produce a palpable distal pulse. A traumatic false aneurysm or arteriovenous anastomosis may even be regarded as nature's way of providing a stream of blood—admittedly diminished but usually adequate—through the main artery to distal parts. Unless the surgeon can improve this arterial supply, he is doing harm by interfering. Sometimes—especially in " four-fifths " division—the main artery may be blocked by clot for a time, but as the clot retracts the main arterial flow is resumed. Surgical intervention may be needed after a few days or weeks, because the enlarging aneurysmal sac presses on the vessels providing collateral circulation or on the main artery distal to its wound, because of suppuration at the periphery of the sac, or because of external hæmorrhage ; but the intervening delay will have provided valuable time for the development of an adequate collateral circulation. In some cases the arterial hæmatoma is large enough to compress the vessels of the potential collateral circulation, and calls for still earlier operative treatment.

C. **Contusion of an Artery**

(1) *With thrombosis.*—The artery appears swollen and discoloured, and feels solid ; pulsation is absent, or feeble. The damage to the arterial wall may subsequently lead to necrosis and, as separation of the necrosed portion may occur, secondary hæmorrhage, internal or external, may follow.

(2) *Without* (*immediate*) *thrombosis.*—The artery—or, more commonly, a patch of the arterial wall—appears discoloured, but pulsation is present. Subsequent necrosis may lead to external secondary hæmorrhage or to the formation of a false aneurysm. At a later date, thrombosis may supervene, and lead to partial or complete occlusion of the lumen ; from such a thrombus, emboli may be detached.

D. **Arterial Spasm**

This is not a common cause of gangrene of a limb. The extent of closure varies ; sometimes a vessel is found completely closed, and so tightly contracted that it resembles a mere string. At other times, the lumen is only slightly narrowed, not sufficiently to interfere with the distal blood supply. The wall of the spastic artery appears " dead white," and the vessel loses both its normal bluish-white appearance and the very fine vascular markings in the adventitia. There may be a local bruise on the vessel wall ; or the vessel may look " thinned," the latter appearance often being indicative of severe damage to its deeper coats. Where there is much bruising in the adventitia, it is rare to find the vessel in spasm—the muscle walls may have been destroyed ; the severely damaged vessel pulsates weakly and thrombosis is a common sequel. Spasm may involve only a short length of vessel (2-3 in.), or it may extend into the peripheral vascular tree. The completely divided vessel has, as a rule, little more than the terminal inch above the division in spasm.

Especially in states of shock, a spastic vessel appears more irritable, and on any attempt to trace the extent of the spasm, increasing lengths of vessel will be found to react in that way. In the upper limb, the distal third of the brachial artery is the most frequent segment to be found in spasm ; rarely does this extend proximal to the insertion of coracobrachialis, although distally the radial and ulnar vessels may be involved. In the lower limb, the lower third of the femoral, the popliteal or the posterior tibial are the usual segments to be involved ; the apex of the femoral triangle is, as a rule, the proximal limit of any spasm. The duration of traumatic spasm varies ; it may last for minutes or for hours ; it may be unremitting and persist even while around it tissues are dying or dead. In spasm of minor degrees, the vessel wall may temporarily relax at long intervals. In the type of spasm which follows a " near miss " of the vessel wall, weak peristaltic waves may sometimes be seen rippling across the contracted segment. It should be noted that trauma that damages the main vessel stream may affect the collaterals in two ways : (*a*) the upper collaterals may be intact but dry, as their mouths are occluded by spasm of the main vessel, and (*b*) the missile responsible for spasm of the main vessel may likewise induce spasm of the collaterals lining or adjacent to its track. Arterial spasm may also follow the prolonged application of a tourniquet, or crush injuries of the extremities, and it has been reported following the concussion effect of mines exploding beneath the feet ; moreover, intense vasoconstriction may be a feature of one phase of traumatic " shock."

E. **Development of Collateral Circulation**

Any considerable reduction of the normal blood flow through a main artery initiates the development of a collateral circulation. This is a gradual process, which extends over periods varying from hours to years. In all varieties of arterial injury except contusion without thrombosis, a collateral circulation begins to develop at once. If it can be ensured that some blood is flowing along the main artery, even for a few days, the collateral circulation developed during that time, although far from perfect, may well be adequate to prevent or reduce the danger of infection or of ischæmic necrosis, especially if the limb be kept cool (p. 18).

A collateral circulation depends first upon existing vascular anastomoses—for example, around joints ; then upon dilatation of the capillary network supplied by branches above and below the arterial lesion, and, later, on the subsequent growth and conversion of these vessels into arteries. This circulation is derived not only from major branches of the artery, but also from numerous unnamed muscular branches, and muscle damage may disrupt a large number of such tributaries. Hence if there has been much destruction of muscle at the level of the arterial lesion—for example, in a gunshot wound fracturing the femur and dividing the main artery—the development of an adequate collateral circulation is unlikely, and primary amputation is advisable. Where only a few muscular branches exist at the level of an arterial injury—for example, in the case of the popliteal artery—an adequate collateral circulation is less likely to develop ; this is one of the reasons why ligation of the popliteal artery has a worse prognosis than that of any other limb artery.

CLINICAL MANIFESTATIONS

A. **Complete Division, Thrombosis or Spasm of a Main Artery**

(1) *Signs and Symptoms.*—Complete interruption of the blood flow in a main artery by division, contusion with thrombosis, arterial spasm or ligature does not necessarily imperil the life of the limb. The collateral circulation can often meet the needs, and evidence of nutritional or functional disturbances

may be fleeting or absent. The final effect of such interruption is determined by a number of factors: (*a*) the site of the vessel injury, and the number of intact and patent collaterals available; (*b*) the extent of the damage to collaterals, for example by a missile; and (*c*) the possibility of dilatation of the collaterals, which is determined in great measure by the general condition of the patient. The radial, the ulnar, the dorsalis pedis, the lower ends of the tibial arteries and the external carotid, are sites which may be ligatured without apparent ill-effects. In contrast, three limb arteries are commonly regarded as dangerous to ligature—the axillary, the common femoral, and especially the popliteal. In the case of the popliteal artery, the risk to the limb is much greater if the injury to the vessel is in the lower part, because consecutive thrombosis may then block the origins of the anterior and posterior tibial arteries. Lesions or ligature of the axillary artery also have a critical level—the subscapular branch; ligature distal to this branch deprives the limb of the abundant scapular collateral flow.

Immediately after the interruption, slowing of the blood in the distal capillary lake gives rise to a state of anoxia which may last for several days. Tissues vary in their susceptibility to oxygen lack, according to their metabolic requirements, which, in turn, vary with their temperature. In a warm environment, blisters form on the ischæmic *skin* within 12 hours, and within 24 hours areas of local necrosis may appear; whereas *muscle* rarely survives more than 6 to 10 hours of total ischæmia. However, if the limb be kept cool, skin may withstand even 48 hours of anoxia, and muscle longer periods than 10 hours. Following sudden total ischæmia, nerve conduction continues for about 15 to 30 minutes, and then ceases; failure of conduction begins distally, gradually extends proximally, and is complete within an hour; conduction may be resumed even after 24 hours of ischæmia, but almost invariably some permanent neural damage remains. The components of a mixed peripheral nerve vary in their resistance to anoxia; nerve endings, motor fibres, fibres subserving touch, temperature, and fast-conducted pain, appear to be particularly susceptible, whereas fibres subserving slow-conducted pain, and vasomotor, sudomotor and pilomotor fibres, are seemingly more tolerant. This variation in susceptibility may be important in producing such sequels as painful conditions of the ischæmic limb.

The first and most striking feature of the acutely ischæmic limb is its deathly **pallor**; every drop of blood appears to have been drained from the skin. This cadaveric pallor—a yellowish-white hue—is more marked if the limb has been elevated previously, or if the environment be cold. But soon the terminal digits become **cyanosed**; the warmer the environment, the earlier does this happen. The local oxygen want causes the fine vessels to dilate; the blood parts with its oxygen; and the warmer the environment the more rapidly does this dissociation of oxyhæmoglobin take place; cyanosis may be evident within a few minutes of the onset of the vascular catastrophe. An increasing diffuse cyanosis now spreads and colours the ischæmic area at first slate-grey, then light mauve, deepening finally to violet; when death of the area is certain, it is purple-black. The extent of the spread of this cyanosis varies. Often the entire limb is affected; at other times, one sees the fingers or toes blue, the hand or foot white, and the forearm or leg cyanosed. Hours later—again depending on the warmth of the environment—**white patches** ("Bier spots") appear and break up the cyanotic areas, and the limb now has a blotchy, mottled look. If the limb be kept cool, it may take days for this blotchy condition to be complete. It is not necessarily an irreversible state, and the limb may recover after it has persisted for a week. If elevation has been maintained, pallor of the limb, rather than cyanosis, may be the predominant feature. In any event, the "Bier spots" spread, diffuse and join, to give the limb a uniform deathly off-white hue. The colour of the limb may be a mixture of cyanosis and pallor; large areas may

become white while the rest remain cyanosed. The white and cyanosed areas now become streaked by irregular sinuous venules—**marbling ;** this is due to the staining by blood of vein-walls which have become porous. It does not signify thrombosis, and recovery of the limb may still take place. It does signify serious vascular insufficiency, usually of more than 24 hours standing. **Blisters,** filled with a sero-sanguinous fluid, now begin to form in the skin, raising it in blebs which brush off easily, leaving an oozing surface. The blisters vary in size, and are an indication of serious skin damage ; nevertheless, at times the damage is found to be very superficial, and when the limb survives, some permanent staining of the skin—or flaking off of a superficial layer of skin—may be the only result of what at one time seemed a catastrophic lesion. Beneath the blisters, or separately, **dark patches** (eschars) may appear ; these are due to damage to the capillaries of the skin, and to blood oozing into the skin-planes ; such dark areas almost invariably die. It is to be noted that in the absence of any but poor means of repair, these dark gangrenous areas in skin may follow the slightest bruising or pressure. As a rule, and particularly in the leg, extensive blistering must be viewed as premonitory evidence of probable extensive gangrene of the limb.

The **cold skin** of the avascular limb is the next most striking clinical feature. With vascular arrest, the skin-temperature falls and, owing to evaporation, it will finally be slightly below room temperature ; the limb **cools** till it feels like that of a corpse. The rapidity of the cooling depends on the temperature of the environment ; in a warm bed the process may take many hours. It is important to realise that the limb does not become suddenly cold. The periphery of the limb cools first, and the digits first show a fall in temperature ; the back of the hand or the foot often remains warm for some time longer. The skin may remain pale, cold and contracted for several days, even when the circulation is recovering. A gradual **creeping warmth,** spreading slowly down the limb, signals recovery, and usually heralds the return of the deeper circulation also ; but it does not necessarily do so, and even when the skin is warm there may be underlying muscle death.

Œdema of the limb is directly consequent on anoxia ; the capillaries become permeable, and plasma or blood may escape into the tissues. This anoxic œdema is most marked where there is still a trickling but inadequate blood supply. Swelling starts at the periphery, in the digits, and often at this stage the cyanosed limb, swollen with œdema, is mistakenly regarded as in a state of venous stasis ; the serious arterial circulatory insufficiency may not be appreciated. The extent and amount of the œdema depend on the height of the limb in relation to the heart-level, œdema being decreased by elevation. Ischæmic muscular palsy reduces the flow of lymph, and pressure on veins by hæmatoma or aneurysm formation may further augment the œdema. It is reasonable to picture this œdema as compressing the capillaries, and therefore in some measure as being responsible for the extent of the ultimate gangrene ; but experimentally it is not the deciding factor, the environmental temperature being the more important consideration. The amount of œdema and the fluid content of the tissues decide whether the gangrene will be **" wet "** or **" dry."** In acute arterial injuries involving a main vessel, gangrene is nearly always wet ; here a massive volume of limb remains loaded with blood at the moment of vascular arrest, and the drying effect of the air is slow. **Infection** of such a gangrenous area is followed by further swelling from inflammatory reaction, but this is possible only if some circulation still exists. Dry gangrene follows gradual arrest of the circulation ; here the gangrenous area is relatively small, and evaporation soon dries it.

Pain is a variable feature. At times, it is agonising, and variously described as burning, a tightening pain, or a feeling as if the limb wants to burst. As a rule, the pain is diffuse ; it may be as severe as any that the human body is

called upon to bear. At other times, the limb feels numb, " terribly cold," or dead ; pins and needles may be complained of. The pain has been shown to be an expression of muscle ischæmia, and it is most severe when the limb has been exercised immediately before or just after the vascular arrest. The pain may come on in paroxysms, lasting for perhaps half an hour, with intervals of freedom of about 2 hours. In the recovering limb, there may be **rest pain** usually due to a local skin lesion or to the involvement of bone or joint. Grossly **exaggerated responses** to painful and thermal stimuli at times follow ligature of main vessels or injury in their neighbourhood ; this is difficult to explain, for in numerous cases such vessels have been ligatured or injured at the same points with no such subsequent effects.

Abrupt vascular arrest of severe degree is invariably followed by gross **disturbance of nerve conduction.** This does not come on immediately, and even after total ischæmia, motor and sensory function may continue for 15 to 30 minutes—the time depending on the warmth of the limb (the warmer the limb, the sooner does conduction cease). Sensory and motor conduction cease almost at the same time. The fact that a limb has been able to function after an injury is a valuable help in excluding associated nerve injury. **Loss of motor power** starts at the periphery ; the intrinsic muscles of hand or foot fail first, and gradually power of movement is lost in the more proximal ischæmic muscles. All muscle reflexes are absent, and the ischæmic muscle does not respond either to faradism or to galvanism. Joints are no longer moved, and exudate accumulates and organises round joint capsules, particularly those of digits ; **stiff joints** and **contractures** may result. **Sensory loss** is often overlooked in the ischæmic limb, especially in a recovering arterial injury. There may be complete anæsthesia or merely hypoæsthesia ; the sensory loss does not conform with any peripheral nerve topography, a helpful feature in distinguishing the arterial paretic lesion from the nerve injury. It is often of stocking or glove distribution. As the vascular supply improves, the anæsthetic area diminishes, and a " slipper " form may be all that remains ; there may also be a tongue-like extension on the anterior and lateral aspects of the leg. Anæsthesia to light touch and to pin-prick are of similar extent, although loss to pin-prick extends to a slightly more proximal level ; often there is a zone of hyperæsthesia at the proximal limit. Anæsthesia on the sole of the foot is easily overlooked, frequently because patients do not complain of it until ulcers develop. **Unpleasant sensory effects** may follow recovery of the circulation : in the areas which were formerly anæsthetic, there may subsequently be burning, formication, tingling or pins and needles. Sensations of heat and cold are often painful. There is a characteristic " slow " pain response ; thus on squeezing, for example, a toe, there is a delay of 1 or 2 seconds before the pain is appreciated ; then the response is one of intense pain and the patient rapidly withdraws his foot ; the pain is described as particularly unpleasant. The recovering muscles are also tender, and even gentle pressure upon them is followed by intense pain ; patients may complain that when walking on irregular surfaces they frequently have to jump aside because of this pain. Rather different effects have been observed in cases where, owing to intense œdema or hæmorrhage, or compression of a limb by tight plaster or tight fascia, the blood supply to nerves has been strangled. Here anæsthesia involving the territory of deep nerves but not that of superficial nerves is characteristic ; a similar slow pain response is also found. It is well to observe that compression pressures as low as 50 mm. of Hg may cause paralysis of nerve trunks, even though the arterial blood supply to the distal limb is not obliterated ; " slow " pain fibres, being resistant to ischæmia, apparently are able to survive. As a general rule, recovery from a severe ischæmic nerve lesion is a slow process, and the final peripheral recovery is usually incomplete. In the leg, the extensor hallucis longus muscle is frequently the last to recover ; the intrinsic muscles of the foot recover slowly, if at all, and the sole of the foot remains anæsthetic.

After ligation of a major vessel, the **distal pulse** disappears immediately. When collateral circulation is well developed, as in an established arterial or arteriovenous aneurysm, the pulse may be fully retained. As a general rule, it takes 4 days in the arm, and 14 days or more in the leg, for the distal pulse to return after ligature of the main artery. It may never return and yet no ischæmic complications may be evident ; especially is this the case after ligation of the external iliac, common femoral, axillary, or subclavian arteries. Again, a feeble pulse may return at a later date, as a result of increasing collateral circulation or perhaps due to re-canalisation of a thrombosed main vessel. Moreover, the pulse wave may be damped down by the clot of a large aneurysm, and an absent or feeble distal pulse in a case of traumatic aneurysm does not necessarily indicate an inadequate collateral circulation. In the acute vascular emergency, in the absence of a local murmur, **persistent absence of pulse** means that the vessel has been divided, has thrombosed, or is in a state of spasm ; except by surgical exposure, there is no way of deciding which has happened. Reliable assessment of the distal pulse is possible only when the limb is warm, for the more superficial vessels, such as the dorsalis pedis or radial arteries, may be obliterated by spasm due to cold. In a fair percentage of normal subjects, the dorsalis pedis pulse is absent ; frequently it is found displaced. The peroneal artery not uncommonly replaces the posterior tibial, and the pulse, when present, is then found anterior to the lateral malleolus. Anomalies of the radial artery are not rare, and the ulnar artery often pursues a superficial subcutaneous course in front of the flexor muscles. The recovering limb should always be carefully inspected for any **abnormal pulsating vessels,** because the presence of such a vessel where normally there is none may be a valuable indication that the collateral circulation is already developing satisfactorily. The presence of a distal pulse does not exclude serious pressure by plaster or splint, sufficient to interrupt the blood supply of the proximal musculature ; it has been shown that the blood supply to muscle and skin may be obliterated by local pressures as low as 50 to 60 mm. Hg.

In arterial injuries the **pulse rate** is usually raised, due mainly to blood loss or to associated pyrexia. A **rising pulse rate,** associated with increasing pain in a limb, may be an important premonitory sign of gas gangrene. During convalescence after a wound, a **persistently raised pulse rate,** especially if it is variable, should raise the suspicion that an arteriovenous aneurysm or aneurysmal varix may be present.

The rapidity of the local return of colour after momentary pressure on the skin is often used as a rough means of estimating the local circulation. It is not a particularly efficient guide, and may be misleading. Where the general circulation is good, the response is good—but a poor response may be obtained with a perfectly adequate circulation. The opposite is also true, and in a dependent limb there may be a quick return and yet a very poor circulation. Cold, and the possibility of local vasoconstriction in traumatic " shock," further diminish the value of the test.

A crude indication of the state of the peripheral circulation may be obtained by placing the limb in a horizontal position, emptying any prominent superficial vein by stroking it, and noting the time taken for the vein to refill. This is very much prolonged in critical circulatory states, and may take as long as a minute. In " shock," peripheral cutaneous vasoconstriction will considerably prolong the time required. If rapid venous refilling be obtained when the main vessel is obstructed, it is an indication that a satisfactory collateral circulation must be reaching the periphery.

The presence of **gangrene** implies irreversible damage to the affected tissues, and in these there can be no improvement. At an early date, it is often difficult to forecast the ultimate extent of tissue recovery after vascular injury : the cold white or blue ischæmic paralysed limb often recovers to an unexpected

extent, and local gangrenous patches in the skin may be the only final manifestations of tissue destruction in a limb where total gangrene at one time seemed inevitable. A digit apparently completely gangrenous often finally sheds merely a superficial skin flake or its nail-bed, the rest of the digit surviving. If there is no improvement in the distal circulation by the third day after a vascular injury, final gangrene of greater or lesser extent may be regarded as certain; **coldness, cyanosis, pallor, absence of distal pulses, mottling** to a violet hue and **blisters** on the skin are the warning signs. As a general rule, by the fifth day the proximal level of gangrene will have been reached and a **line of demarcation** will be evident. This does not change, as is commonly supposed, in the traumatic type of case. Considerable septic absorption may take place at the line of demarcation. Certain areas of skin appear to be particularly sensitive to ischæmia—the distal phalanges, the middle 3 toes of the foot, the heel and the area over the heads of the metatarsals. The extent of gangrene in a limb will depend on the *site* of the vascular lesion. When the axilla is injured, if the limb dies a line of demarcation develops in the middle of the forearm. Damage to the brachial artery in its upper part (usually associated with much local tissue damage) is followed by a line of demarcation through the metacarpal bones. The collateral blood supply of the thigh is so abundant that ischæmic gangrene above the knee joint is rarely seen, though severe wounds of the thigh and buttock carry a special risk of gas gangrene. In lesions of the superficial femoral and popliteal arteries the line of demarcation in ischæmic gangrene is through the middle of the leg; with injury of the proximal parts of the posterior tibial vessels, the line of demarcation usually passes through the metatarsal bones. The amount of local destruction of muscle, and thus of collateral vessels, determines in great measure the extent of the gangrene. Thus, if, in gunshot injuries producing fracture of the femur or wounding the knee joint, the femoral or popliteal artery be damaged at the same time, gangrene of the distal part of the leg almost invariably follows.

The effects of ligature of the **internal carotid artery** must now be considered. The ultimate effects depend in great measure upon the pre-operative condition of the patient as regards exsanguination, state of consciousness, paralysis, etc., as well as upon the variable anatomy of the circle of Willis. Signs of **cerebral anoxæmia** may be absent or transient. In other cases the effects may be serious; immediately, or sometimes after a lapse of some hours, **hemiparesis** becomes evident; a left-sided lesion may be complicated by **aphasia.** However, the paresis may not be severe, and may show itself merely by slight weakness of the contralateral arm, or by clumsy movements of the thumb and fingers. The cerebral ischæmia may be followed by attacks of **dizziness** and **faintness,** which may end in **coma.** Some patients develop **epileptiform attacks,** said to be due to the ischæmic area acting as an irritative focus. **Inability to concentrate, difficulty in remembering words,** and even **dementia** may be predominating features in the picture of cerebral ischæmia.

(2) *Complications.*—It should always be borne in mind that, in a seriously ischæmic limb, even a mere contamination of a muscle wound with an anærobic organism carries a grave risk of fulminating **anaerobic infection,** which may cause the death of the patient within 6 hours of his injury. For this reason, many limbs have to be amputated before the amount and degree of ischæmic necrosis have become apparent. In such cases, the recommended prophylactic measures against gas gangrene should always be applied*. Similarly, **aerobic infection** is much more likely to occur, and its spread to be more rapid, in an ischæmic limb than in a limb with a normal blood supply.

There is now strong evidence that **Volkmann's contracture** follows a purely arterial lesion. The ischæmic muscle dies and is replaced by fibrous tissue, which scars and contracts to produce typical deformities. There can be no

* The latest recommendations under this heading are given in *Army Med. Dept. Bull. Supp. No.* 15, 1944, reprinted in full in *Bull. War Med.*, 1944, Aug., v. 4, No. 12, 687–90.

recovery of such muscle, and the ultimate prognosis for the limb depends on which muscles have survived; this, in turn, will depend on the collateral circulation. In the forearm, the extensor group of muscles, and frequently the brachioradialis, will survive. In the leg, the gastrocnemius and the upper part of the soleus often are found to be unaffected, although the remainder of the muscles of the calf have suffered; this is because the blood supply to the muscles named comes off high in the popliteal fossa and has escaped. The development of the Volkmann's state can be accurately diagnosed by muscle biopsy, which shows islands of dead muscle, devoid of nuclei and surrounded by zones of cellular activity. *Muscle biopsies with this object should not be taken early after vascular injuries.* In the leg, the woody mass of ischæmic muscle associated with the Volkmann's state induces a most disabling contracture; the condition is frequently missed at first and usually leads to amputation. Occasionally, after ligature of the femoral artery, the only evidence of distal damage is a colliquefactive necrosis of the anterior tibial group of muscles. A soft and fluctuating swelling, without heat or signs of inflammation, can be felt, and on incision it exudes a grumous fluid. A number of cases has been described in which, many years after femoral ligature, radiographs have shown the anterior tibial muscle mass to be completely calcified; this condition may be a late stage of muscle infarct.

Causalgia rarely follows a pure arterial injury; there are usually associated partial wounds of the median or sciatic nerves. The intense burning pain, the extreme hyperalgesia to the lightest touch—even to a current of air or to warmth—make the diagnosis clear. It is important to recognise causalgia as a possible complication of a partial ischæmic state. At times the condition is much improved by sympathectomy, but the results of this operation are variable.

(3) *Psychological Aspects.*—In war, patients readily adjust themselves to amputation after the development of gangrene, and commonly face it with equanimity. There may, however, be marked **restlessness** in the early stages of arterial injury, because of pain (p. 9) or loss of blood; where the loss of blood has been extreme, the restlessness may be almost uncontrollable until the deficit has been made good by adequate blood transfusion.

B. Partial Division of a Main Artery

The clinical manifestations of partial division differ from those of other arterial injuries in that, since some blood is still flowing through the main artery, there will be no immediate evidence of ischæmia, though, after the lapse of several days or a few weeks, gradual compression of the collateral vessels by an enlarging aneurysmal sac may produce a slowly increasing impairment of the distal blood supply. There is often a history of **undue bleeding** relative to the size of the skin wound, and of **undue local swelling** relative to the number of hours that have elapsed since wounding. (Local swelling may be absent in aneurysmal varices, where the injured artery and vein communicate directly, without the intervention or association of a false sac (p. 6)). The **distal pulse** is usually present, though its volume may be diminished; sometimes it is temporarily absent. A persistent unexplained **tachycardia** is suggestive (see also p. 11). **Local pulsation** is often absent in the early stages, and if present is generally difficult to detect. The most constant sign is the presence of a **short systolic murmur.** The ideal of auscultating all wounds may not be possible in practice, but *any artery that might pass near a wound track should be examined with a stethoscope,* especially if the signs mentioned above are present. It must be remembered that many arterial wounds are produced by very small projectiles, and in patients with multiple small wounds, the main arteries should be auscultated as a routine. The murmur usually appears within a few hours of the injury, though occasionally its onset may be delayed for several days. At first, it is a faint short systolic whiff, heard

best near entry or exit wound, or where the artery is closest to the surface, and it is not conducted. Later, it becomes louder, and is usually conducted further peripherally than centrally, though in arteriovenous fistulæ the reverse occurs; with a false aneurysm, it remains permanently of the partial-cycle systolic type, but it becomes of the complete-cycle type, with systolic accentuation, in an arteriovenous fistula. In the head and neck the murmur may be audible to the patient. Arteriovenous fistulæ produce the loudest murmurs, and often a **thrill** can be felt. In a few cases, especially in arterial wounds of the lower extremity, a faint **cardiac systolic murmur** may be heard. There is commonly a **fall of blood pressure** in the distal vessel, which often amounts to 10 to 20 mm. Hg, both systolic and diastolic. The diastolic fall is greatest in cases of arteriovenous fistula, though its extent depends on the size of the vessel and on that of the arterial leak. It is well to remember that normally the blood pressure is higher in the lower extremity than in the upper. The amplitude of the excursions of the mercury column—at readings 10 to 20 mm. Hg below the systolic pressure—gives some indication of the combined amounts of blood reaching the limb through the main artery and the collateral circulation. However, the extent of the pulsations may be affected by swelling or by an excess of fatty tissue.

PRINCIPLES OF TREATMENT

A. Control of Hæmorrhage in the Forward Areas:—

Four methods are available, the order of preference being: (*a*) local pressure, (*b*) application of forceps, left *in situ*, (*c*) ligature, and (*d*) tourniquet. The need to use a tourniquet should be regarded as a confession of failure, and it should be applied only as a last resort.

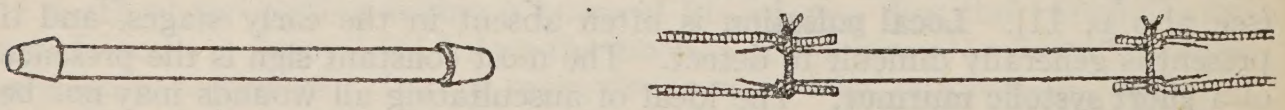
B. Treatment at Centres where Adequate Facilities Exist and where Patients can be Retained:—

(1) Complete Division of a Main Artery.

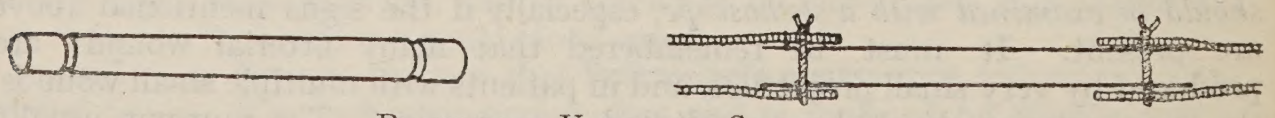
(*a*) *Axial anastomosis.*—The ideal of axial anastomosis in the treatment of a completely severed artery is rarely advisable or even possible in high velocity projectile wounds, because, owing to the gap produced by the excision of the damaged arterial ends, the anastomosis usually cannot be effected without tension, if at all. Moreover, sepsis, resulting from the initial contamination of the wound, is very liable to lead to breakdown of the suture line. These arguments do not apply to stab wounds, in which contamination is usually slight and there is no necessity to excise the arterial ends: axial anastomosis —if possible, direct end-to-end anastomosis (Appendix A, p. 20)—should be attempted here. Since thrombosis is the commonest cause of failure, anti-coagulants (e.g., heparin) should be employed for 7 to 10 days (for the technique of administering heparin, see Appendix B, p. 22).

(*b*) *Artificial tubes.*—There are two main types of single metallic tube for bridging gaps between the vessel ends in the axial anastomosis of arteries: these are the Tuffier silver cannula, and a vitallium cannula, as made for Blakemore and his colleagues (Fig. 2). Such a tube is inserted into the proximal and distal ends of the severed artery, which are tied over it. In the war of

Fig. 2



Tuffier Silver Cannula



Blakemore Vitallium Cannula

(See Blakemore, Lord and Stefco, *Surgery*, 1942, **12**, 488-508. The use of free vein grafts with a vitallium tube, or with two vitallium cuffs, to bridge severed arteries, as also described in this paper, and in others by the same authors, is not considered in the present Memorandum. It has given promising results in dogs, but its clinical possibilities have not yet been sufficiently explored for it to be recommended for trial elsewhere than in special Vascular Injuries C ntres.)

1914-18, Tuffier's cannula usually failed in its purpose, because thrombosis occurred within a few hours ; the use of heparin, however, should prevent this. Nevertheless, the axial anastomosis attainable by the use of single metallic tubes in direct contact with the bloodstream is necessarily only temporary. The tied ends of the artery necrose, and leakage takes place about the fifth day ; the tube should therefore be removed not later than the third or fourth day, both ends of the artery having first been ligated and the heparin discontinued. Though clearly not ideal, this method provides a nearly normal blood supply for 3 or 4 days, and this time is valuable for increasing the amount of the collateral circulation, and diminishing the risk of infection and ischæmic gangrene. To stimulate the formation of the collateral circulation, a tube slightly smaller in diameter than the lumen of the artery should be used.

In an attempt to provide a permanent bridge, lined with intima, for the axial repair of severed arteries, various workers have experimented with the use of free vein grafts (see, for example, the caption to Fig. 2). These methods involve the use of heparin ; they are considered unsafe for use in heavily contaminated wounds, and are likely to be applicable only in special Vascular Injuries Centres ; they will therefore not be discussed further here.

(*c*) *Ligation of proximal and distal ends.*—If the above methods for the axial repair of completely severed arteries are not practicable, and they seldom are, both ends of the divided artery must be ligated. It must be realised that in many cases the limb, especially if it is the lower extremity, will shortly require amputation on account of the rapid spread of infection or the establishment of ischæmic gangrene.

Although there is some difference of opinion as to whether or not the main vein should be tied if a main artery has to be ligated, the majority view at present is that the vein should be tied.

(2) Partial Division of an Artery.

(*a*) *When the entry and/or exit wounds in the soft tissues are small.—Expectant treatment.*—**Clinical evidence of a lateral wound in an artery should be a definite contra-indication to primary excision of the wound of the soft tissues :** such cases should be watched, and although some may require surgical intervention after the lapse of some days or weeks—on account of suppuration, rapid increase in the size of the aneurysmal sac (pressing on the collateral circulation or on the part of the main artery distal to its wound), or because of external hæmorrhage—valuable time will have been gained for the development of a collateral circulation adequate for the control of infection. This time is usually far in excess of that possible with a Tuffier tube. Late surgical treatment should consist of immediate proximal and distal ligature, opening the sac and ligating any branch opening into it.

Should the surgeon fail to diagnose this type of arterial injury, and be confronted with a lateral wound in an artery during the primary excision of the wound in the soft tissues, one of the procedures outlined under (*b*) below should be attempted, preferably (iii).

(*b*) *When the entry and/or exit wounds in the soft tissues are large.*—It is not often that patients with this type of wound reach a properly equipped centre without previous surgical treatment, because the persistent external hæmorrhage will generally have necessitated the application of forceps or ligature in the forward area. What is common is that the surgeon, after failing to recognise clinically a lateral wound in an artery, has, by performing primary excision of a small wound in the soft tissues, converted the case into one of this type, and during the operation is suddenly confronted with a lateral hole in a main artery. One of the following procedures should then be adopted :—

(i) *Suture of the arterial wound.*—Unfortunately, this is not often advisable, since the opening in the arterial wall resulting from the essential excision of the edges necessitates a longitudinal suture line which is apt to lead to extreme narrowing or even to obliteration of the lumen (see Figs. 1 and 3, pp. 5 and 20). Suture may be possible in the case of a small wound in a large vessel, or in many lateral wounds produced by a stab, where there is no necessity to excise the edges of the arterial wound, although the adventitia in the neighbourhood should be removed. For the technique of arterial suture, see Appendix A (p. 19). Heparin should be given (Appendix B).

(ii) *Axial anastomosis.*—From what has been said under (1), (*a*), it will be clear that this procedure is rarely advisable.

(iii) *Making a musculo-fascial barrier over the arterial opening.*—This is an attempt to imitate nature, by providing a barrier to the external escape of blood, with the definite object of allowing a false aneurysm to develop and thereby securing an adequate blood flow through the main artery. One or two layers of fascia or muscle should be sutured over the wounded vessel. Heparin must not be used in such cases.

(iv) *Artificial tubes.*—The use of these tubes has been described under (1), (*b*). It must be remembered that the use of an artificial tube (without vein graft) provides for the main arterial blood flow for, at the most, 4 days, while the method suggested under (iii) above, will usually do so for at least a week, and often for several weeks.

(v) *Ligation of the artery immediately above and below, after division.* This procedure is a last resort, as mentioned under (1), (*c*). The main vein should probably also be tied in such cases.

(3) Contusion.

(*a*) *With Thrombosis.*

Owing to the length of the damaged portion, and the necessity for excising the whole of it, axial anastomosis is never possible. The following methods of treatment are available :—

(i) *Incision and removal of clot.*—The artery is incised above the damaged portion, the clot extracted and the wound sutured. The administration of heparin (Appendix B) is a necessary adjunct to this procedure. As the damaged arterial wall may subsequently necrose, a layer or layers of muscle or fascia should be sutured over it ; where this is not possible, the artery must be divided between ligatures, not later than the third or fourth day, and the damaged segment resected.

(ii) *Artificial tubes.*—The use of these has already been described under (1), (*b*).

(iii) *Ligation of proximal and distal ends after excising the damaged portion ;* see (1), (*c*).

(*b*) *Without Thrombosis.*

(i) *Making a barrier of muscle or fascia.*—As the damaged part of the arterial wall may subsequently undergo necrosis, a barrier of muscle or fascia should be made, as already described, to prevent external hæmorrhage, and to ensure the formation of a false aneurysm. Heparin should not be used.

(ii) *Ligation of proximal and distal ends after division.*—If (i) is not possible, the artery must be divided, and both ends ligated, not later than the third or fourth day, as a precaution against external secondary hæmorrhage ; any damaged segment should be resected. The main vein should probably also be tied.

(4) Arterial Spasm.

The spasm may be discovered accidentally, in the course of vessel exposure during wound excision ; or it may be discovered because absence of pulse has suggested division of an artery, thrombosis, or occlusion by a fractured bone. Spasm is rarely tight—some blood still oozes through—and it nearly always passes off within 24 hours ; the pulse may return feebly at first, and then become stronger later. Unless the collaterals are extensively damaged, by itself spasm rarely determines the fate of the limb. The vessel in spasm should be cleared gently of the local blood clot, and warm saline applied. Local injection of procaine has been tried, but reports indicate that it is of no value. Peri-arterial sympathetic stripping should not be done ; reports of cases show that it has seldom been successful and that it may increase the spasm. Excision of a segment of the vessel is not advisable unless it be extensively bruised. Further information is needed on the effect on the spasm of sympathetic procaine block ; so far, clinical reports have not been encouraging. If the spasm does not relax after the vessel has been simply cleared, it is best to leave the vessel alone, and to rely on its own natural recovery. Where there is much collateral damage, restoration of the main vessel stream may be more urgent—and it may be advisable to dilate forcibly the contracted segment by the proximal injection of isotonic saline.

MANAGEMENT OF THE ISCHÆMIC LIMB

It should be emphasised at the outset that the ischæmic limb needs **constant observation** like the acute abdomen : it should not be left unwatched for the night " to see what it is like in the morning."

A. Attention to Patient's General Condition

This is the first consideration, because the onset of gangrene after vascular damage is often determined by the condition of the patient. Strong peripheral vasoconstriction, including the major vessels, is maintained while the general condition is poor. This rationing of the blood flow to the limbs is more strict where wound "shock" has followed hæmorrhage or trauma. The administration of adequate quantities of fluid is a fundamental part of resuscitation. **Blood transfusions,** early, rapidly, and in massive amounts, are essential. Blood is to be preferred to plasma or serum in the early stages after hæmorrhage ; it is important to restore the highest possible oxygen-carrying-power to the trickling stream in the ischæmic limb. Resuscitative measures may need to be hurried ; with extreme exsanguination, a transfusion rate of a pint in 5 minutes or less (given under pressure—with a Higginson syringe) may be necessary to save life. Speed is essential also because striated muscle is particularly sensitive to anoxia, and muscle death will provide the particular opportunities required for the invasion of the tissues by gas gangrene organisms present in the wound. Three to 4 pints of blood is an average amount needed in the bleeding wound-shocked patient ; it is advisable to give whole blood in amount equivalent to the estimated blood loss, then plasma or serum as necessary. It must not be forgotten that with any infection, and particularly with gas gangrene, extensive œdema formation rapidly depletes the general circulation. Additional blood, plasma or serum in considerable amounts may thus be called for, to restore the blood volume and serum protein content.

Oxygen therapy through a B.L.B. mask produces a slight but significant increase in the oxygen content of arterial blood. Given intermittently for short periods, it may thus be of advantage not only from the point of view of the general condition, but also for the local state of the ischæmic limb. Its value in the treatment of traumatic "shock"—apart from the presence of complications such as pulmonary œdema and chest wounds—is doubtful (see also " The Treatment of ' Wound Shock '," *M.R.C. War Memorandum No.* 1. Revised edition, 1944).

B. **Reduction of Metabolic Needs**

(1) *Rest.*—The oxygen demands of resting tissues are small. The limb should not be allowed to wallow unsupported in the bed; **immobilisation** will limit infection and septic absorption, and will keep the temperature down and lessen the metabolic needs. The limb should be supported preferably on a Thomas splint, or on well-padded Cramer wire. **Morphine** should be given in regular doses; it allays restlessness and anxiety. It is preferably given slowly into the infusion fluid, in amounts not exceeding gr. $\frac{1}{6}$ four-hourly; in a shocked patient, subcutaneous injections are poorly absorbed. **Phenobarbitone** is also of value, because it promotes sleep. It is often forgotten that over-fatigue, fear and noise are powerful vasoconstricting stimuli. The reassuring word may itself do much to ease the anxious restlessness.

(2) *Local cooling.*—**The limb should be kept cool.**—By suitable cooling, cell metabolism is much reduced and very little oxygen is thus needed. Life in the muscles and other tissues can thus be maintained whilst the collateral circulation is being established and repaired. Extreme cooling, such as can be produced by the application of ice to the limb, is inadvisable, as very low temperatures damage the skin. The limb, lightly wrapped in sterile cotton wool, should be kept exposed under a cradle—and, if the room or climate be warm—ice bags should be suspended from the cradle, or a fan blower used to cool the surrounding air.

C. **Assistance of the Collateral Circulation**

(1) *By avoiding compression of the collateral vessels.*—**Plaster treatment is contraindicated** if there is any doubt about the efficiency of the vascular supply to the limb—the collaterals may be easily compressed. When a limb is already in plaster, it is better to remove the casing completely; splitting the plaster is not a sufficient safeguard, and often merely leads to bulging œdema and local pressure necrosis. The position of the limb should be such that pressure is not allowed to fall on the side of the establishing collateral vessels.

Massage or manipulation should generally be avoided—it may add to the injury. Nevertheless, **the joints, especially the smaller joints, of the ischæmic paralysed limb, should be passively moved through their entire range of movement at least twice a day;** otherwise, pericapsular organisation takes place, with much subsequent stiffness and contracture. Where there is a fracture, gentle traction may be necessary, for the vessel may sometimes be trapped between the bone ends.

Incisions of the deep fascia, to relieve the compression of the collateral circulation caused by an increasing arterial hæmatoma or an extravasation of blood from some small artery or a vein, may become urgently necessary, especially in the calf and popliteal regions. In the case of an arterial hæmatoma, the incision should be separate from the original wound, to avoid the risk of abolishing the protective barrier of clot and fascia which alone prevents the rupture of the arterial hæmatoma externally.

Elevation of the limb, as already indicated at p. 9, limits œdema, which may compress the developing capillaries. Œdema is directly consequent on the anoxia, and is exacerbated by failure of lymph flow, due to the muscle palsy that follows any severe degree of ischæmia. It comes on rapidly, and elevation, to be effective, must be done early; even during transport from the operating theatre, attention must be paid to this. The height of elevation desirable is open to argument; the comfort of the patient is often a good guide—he will complain of pain if the elevation is excessive. As a general rule, the limb should not be elevated much above heart-level.

(2) *By measures to "force" the collateral circulation; and the use of vasodilator drugs.*—**Warming the rest of the body, whilst keeping the ischæmic limb cool,** in an attempt to force the collateral circulation, is a measure now being tried with reported encouraging results. The principal sites of vasodilatation by such heating are the hands and the feet, the increased blood flow to the limb muscles being negligible. The method finds its particular application, therefore, in the attempted vascularisation of cutaneous areas, such as the fingers and toes. As skin can stand 24 hours of ischæmia, providing it is kept cool, heating of unaffected parts of the body may be delayed until the general condition is satisfactory and it is certain that the critical period of muscle survival has passed. The body may be warmed either by hot water bottles, hot drinks, radiant heat, or by immersion of unaffected limbs in hot water at 110° F. It is reasonable to start such heating, say 12 hours after the vascular catastrophe. When the patient is already febrile, and during sleep, such warming is not needed.

The value of **sympathetic denervation** is not yet decided. Encouraging reports have been received, especially of its worth in the arterial or arteriovenous aneurysm type of case. In the acute type of injury, with the patient severely ill, sympathectomy is too formidable a procedure, and is rarely justifiable. Sympathetic block by procaine, or procaine in oil (p. 22), is, however, a measure that is simple and could be easily practised in cases of limb ischæmia due to vascular wounds. As indicated on p. 17, its value is doubtful in cases of pure arterial spasm.

Smoking should not be allowed: it may cause peripheral vasospasm. **Vasodilator drugs** have not proved very effective, but papaverine hydrochloride gr. ½ intravenously two-hourly, may relieve the local ischæmia. A deep, thorough sleep is the most efficient natural vasodilator; phenobarbitone in regular doses is here of special value. Alcohol may also be useful for its vasodilator and sedative effects.

It has been demonstrated that **hypertonic saline** given intravenously will cause an increase in the muscle and skin circulation. Two hundred c.c. of a 2 per cent. solution have been given in base hospitals with encouraging results.

D. **Prevention of Infection**

Careful asepsis is essential in the repeated examinations of the devitalised limb. Small incisions to test viability, and early muscle biopsies are to be condemned (p. 13). Masks should be worn, and particular care taken in the examination of any adjacent wounds. The entire limb should be shaved, washed with soap and water, and dried with a sterile towel; and a sulphonamide or other bacteriostatic powder should be dusted on the wounds.

After the acute phase has passed, and the collateral circulation has so far progressed as to give rise to a reasonably quick capillary return, a naturally warm limb and a perceptible distal pulse, **active muscular movements** are of help in promoting the collateral circulation still further.

APPENDIX A

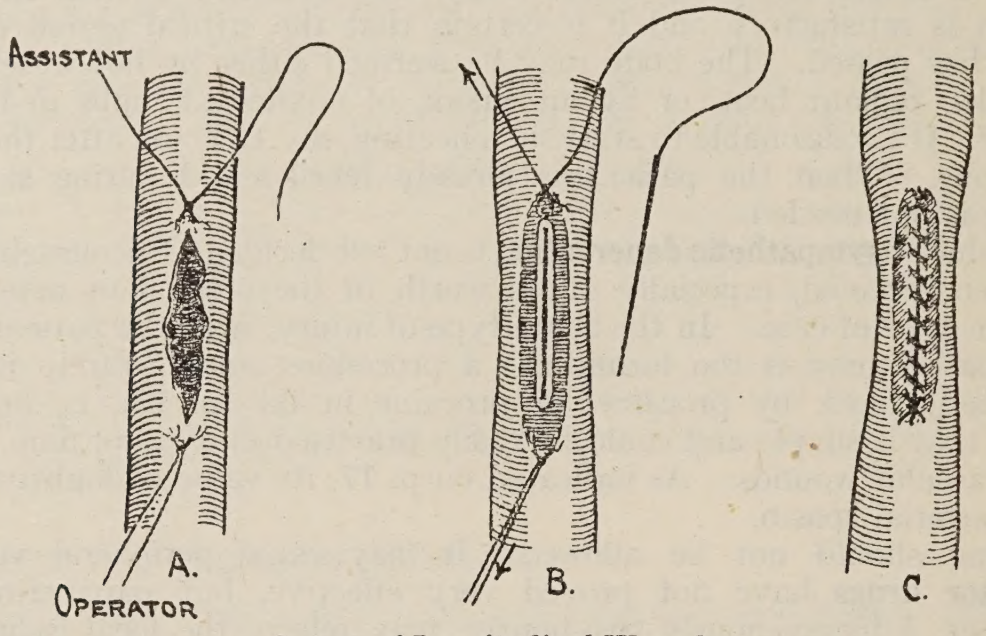
Technique of Arterial Suture

Special instruments required.—Small fine-pointed toothless dissecting forceps; straight mosquito forceps (6), one for use as needle-holder; bulldog clamps for occluding vessels; straight sharp iris or cuticle scissors; arterial sutures on eyeless needles, straight and curved, No. 2/0 (T) black braided silk, out of sterile liquid paraffin; sterile isotonic (3·8 per cent.) sodium citrate solution; pipettes. In all cases, the circulation in the whole limb is controlled; or the circulation in the wounded artery is controlled above and below the lesion, by bulldog clamps or by tapes tied temporarily over a short piece of rubber tubing.

Longitudinal cuts.—The adventitia is delicately removed from the immediate neighbourhood of the wound. The edges of the wound are trimmed evenly, but sparingly. The interior of the artery is cleansed with sterile isotonic (3·8 per cent.) sodium citrate solution, and kept moist with either sterile liquid paraffin or heparin solution.

A stay suture is inserted a few millimetres beyond each end of the wound (Fig. 3 (A, B)), the needle being retained on the stay farthest from the operator.

FIG. 3



Suture of Longitudinal Wound.

With his left hand, the operator puts tension on the stay suture nearer to him, while the assistant deals similarly with the other stay; the wound edges are thus raised and everted. The surgeon sews towards him: a straight needle is usually most convenient. A simple continuous stitch is used (Fig. 3 (C)); on the surgeon's side it is carried beyond the wound, and it is ended by tying the suture to the stay. The circulation is gradually restored, either by the slow release of the tourniquet or by releasing first the distal and then the proximal bulldog clamp or temporary ligature. Gentle pressure is made on the suture line by gauze moistened with warm saline; any persistent oozing point is dealt with by inserting an additional stitch, care being taken that the original continuous stitch is not divided by the re-insertion of the needle. When hæmostasis is secure, and not till then, the stays are cut short.

Transverse wounds.—These are sutured like longitudinal wounds when their length does not exceed one-third the circumference of the artery. Longer transverse wounds should be treated by completing the division of the vessel, and performing end-to-end anastomosis if possible.

End-to-end anastomosis (Fig. 4).—This procedure can be adopted only when the divided ends, after trimming, will come together without tension, and without the sacrifice of neighbouring collateral vessels.

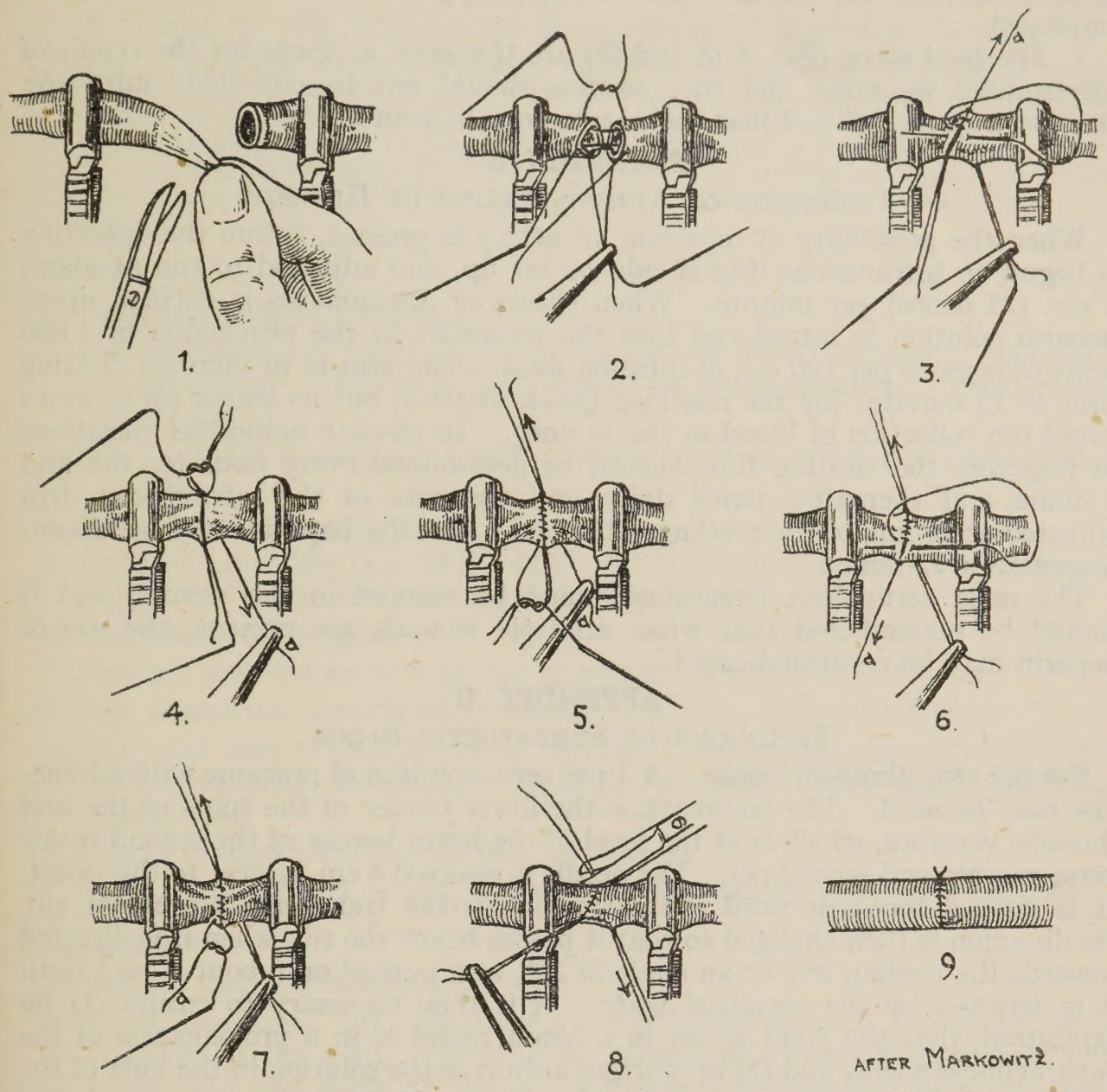
1. The circulation is controlled.

2. The area of operation is isolated by gauze moistened with warm saline.

3. The ends of the divided vessel are trimmed squarely by sharp scissors, a sharp knife, or a safety-razor blade held in artery forceps.

4. The adventitia is cleared from the neighbourhood of the suture line, by drawing it over each cut end, between the thumb and forefinger, cutting it

FIG. 4



Anastomosis of Transected Vessel.

level with the trimmed ends (Fig. 4 (1)), and allowing it to retract. This is a most important step.

5. The interior of both proximal and distal segments is cleansed with sterile isotonic sodium citrate solution dropped from a pipette, and is moistened with sterile liquid paraffin or heparin solution.

6. Three stay sutures are first inserted, which triangulate the ends of the vessels (Fig. 4 (2)) ; the first is placed posteriorly. Each suture is tied (with a surgeon's knot). The needle is retained on the first stay suture, the short end of which is caught in a mosquito forceps. The other stay sutures are caught in forceps, about 10 cm. from the vessel. Each side of the triangle is sewn in turn, beginning with the side from the posterior stay to the anterior stay farthest from the operator (Fig. 4 (3)). The surgeon sews towards him, the stitches passing through all coats of the vessel, about 2 mm. apart. The continuous stitch is tied to each stay suture on reaching it—the other sides of the triangle being presented in turn to the operator, by changing the stays on which tension is made (Fig. 4 (4–7)). A continuous over-and-over stitch is used. It is highly important that uniform tension and a slight " lift " be maintained on the two stay sutures bounding the side which is being sewn. In the case of the carotid artery, which after division tends to remain circular

in section, two stay sutures diametrically opposite to each other may be employed.

7. The final steps (Fig. 4 (8 and 9)) are the same as those for the repair of longitudinal wounds; the stay sutures should not be cut short until the surgeon is quite satisfied that the anastomosis is secure.

APPENDIX B

Technique of Administration of Heparin

When the possibility of repairing an artery is present, before the operation is begun an intravenous drip should be set up, and adjusted to run at about 1 c.c. (25 drops) per minute. When suture or anastomosis is decided upon, heparin solution is introduced into the reservoir, in the proportion of 1,000 units of heparin per 100 c.c. of infusion fluid. The aim is to increase clotting time to 15 minutes (by the modified Quick method) but no longer (in order to avoid the collection of blood in the wound). To obviate individual variations in response, the clotting time should be determined every hour for the first 4 hours, and thereafter twice daily, and the rate of the intravenous drip adjusted according to the readings obtained. Usually heparin may be discontinued after 72 hours.

The most scrupulous hæmostasis must be secured in the wound, and it should be remembered that when multiple wounds are present, the use of heparin may be contraindicated.

APPENDIX C

Technique of Sympathetic Block

For the arm (thoracic block).—A 1 per cent. solution of procaine with adrenaline may be used. The landmark is the lower border of the spine of the first thoracic vertebra, which is at the level of the lower border of the second transverse process and second rib. The needle is inserted 4 cm. lateral to this point. It is pushed forwards until it is arrested by the transverse process or rib. Its direction is then changed so that it passes below the rib, when it is directed towards the median line at an angle of 20°, and pushed on (about 3 cm.) until it is arrested by the vertebral body. It is then necessary to ensure (1) by aspiration, that the point is not in a blood vessel or in a prolongation of the subarachnoid space, and (2) by putting a drop of the solution in the butt of the needle, and observing that it is not aspirated, that the point is not in the pleural cavity. When the point has been correctly placed, 20 c.c. of the solution are injected slowly.

For the leg (lumbar block).—The simplest method of blocking the lumbar sympathetic trunk is by the injection of a comparatively large volume of 1 per cent. procaine-adrenaline solution into the connective tissue antero-lateral to the third lumbar vertebral body.

The needle should be 10 to 12 cm. long, and is introduced at an angle of about 45° to the sagittal plane, at a point 7 cm. (4 fingers' breadth) from the mid-line, at the level of the interval between the second and third lumbar spinous processes. It is passed medially till it meets the lateral aspect of the vertebral body, when it must be withdrawn sufficiently to permit of its being reintroduced more and more anteriorly till it just makes contact with the antero-lateral aspect of the vertebral body yet can slide on past it. The needle point should be moved on 1 cm. beyond the point of contact with the bone, and then (after withdrawing the plunger to make sure that a vessel has not been entered) 30 c.c. of the procaine-adrenaline solution are injected.

When correctly placed, the solution will pass up and down for a considerable distance along the inner border of the psoas muscle, and will thus bathe sufficient of the lumbar sympathetic trunk to produce vasomotor paralysis in the leg vessels for a few (up to 6) hours. A solution of procaine in oil will produce a similar but more prolonged effect.

APPENDIX D

A Guide to the Writing of Case Notes in Vascular Injuries

(Note.—*This Guide was prepared by the Sub-Committee primarily for use at the special Vascular Injuries Centres in Britain. It is realised that a number of the laboratory and instrumental investigations proposed would involve the co-operation of trained experts, and the use of elaborate equipment, not generally available at hospitals in the forward areas. Nevertheless, it is desirable that the suggestions for history taking, clinical examination, operation notes, etc., should be followed by all surgeons dealing with vascular injuries, in so far as may be practicable.*)

History :

1. Date and hour of wounding.
2. Circumstances of wounding : in battle, in civil air-raid ; exposure ; exhaustion ; likelihood and degree of contamination ; compression injury.
3. Site(s) of skin wound(s) : large or small ; with or without retained foreign body.
4. Posture when wounded.
5. Type of projectile (e.g. H.E., M.G., etc.) if known ; or of other weapon (e.g. bayonet), or missile (e.g. glass).
6. Immediate sensations.
7. Amount and nature of external bleeding. Method of arrest.
8. Any immediate associated features : fractures, burns, paralyses.
9. Date and time of recognition of vascular lesion : time of onset and course of symptoms and signs : swelling, murmur, thrill, pain ; tinnitus, proptosis.
10. Course of associated lesions (e.g. paralyses).
11. Previous treatment, if any, including indications for procedure adopted.

Examination :

1. General physical examination.
2. Blood pressures : (*a*) systematic ; (*b*) if lesion is in an extremity, or in course of the main vessels to an extremity, record B.P. proximal (if possible) and distal to lesion ; compare corresponding limb on sound side.
3. Local lesion—name of vessel(s) involved :
   Size of artery, proximal and distal ; presence or absence of peripheral pulses ; venous dilatation and pulsation ; extent.
   Murmur : nature, duration, conduction (central and peripheral), audibility.
   Thrill : duration, conduction (central and peripheral).
4. Pulse rate (charted). Effect of obliterating fistula if present, on pulse rate and B.P.
5. Nutritional lesions : colour of skin of extremity (pale, cyanosed, mottled) ; effect of posture on this. Atrophy—hypertrophy—measurements of limbs. Oedema. Ulceration. Gangrene.
6. Associated lesions : state of muscles, tendons, nerves.
7. If carotid system affected, neurological and psychological examinations.
8. Heart : size, efficiency. Murmur(s)—duration, conduction.
9. Evidence of aerobic and/or anaerobic infection.

Special Examinations :

1. Electrocardiogram.
2. X-ray for size of heart.

3. X-ray for pulmonary congestion.
4. X-ray fistula—foreign bodies, calcification.
5. Blood : count, haematocrit. Venous oxygen tensions, normal and beyond fistula. Blood volume.
6. Oscillometry.
7. Skin temperature readings.
8. Arteriogram.
9. Polygraph tracings.

} If possible.

Operation :

1. Indications for operation.
2. Exposure : vessels involved—nature, size, arrangement.
3. Associated lesions (e.g. of nerves).
4. Operative procedure.

Post-Operative Course :

1. Repeat B.P.s. Date and time of return of pulses : pulse chart. X-rays for size of heart and pulmonary congestion.
2. State of limb. ? Sensory loss.
3. Repeat blood examinations.
4. If in carotid system, repeat neurological and psychological examinations.
5. Repeat electrocardiogram.

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